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Yuki Nishiguchi, Keisuke Takano, Yoshihiko Tanno

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The Need for Cognition Mediates and Moderates the Association between Depressive Symptoms and Impaired Effortful Control

Authors
Yuki Nishiguchi[a]*, Keisuke Takano[b,c], Yoshihiko Tanno[a]

[a]Graduate School of Arts and Sciences, The University of Tokyo, 3-8-1 Komaba, Meguro-ku, Tokyo 153-8902, Japan

[b]College of Humanities and Sciences, Nihon University, 3-25-40 Sakurajosui, Setagaya-Ku, Tokyo 156-8550, Japan

[c]Center for the Psychology of Learning and Experimental Psychopathology, University of Leuven, Tiensestraat 102, box 3712, 3000 Leuven, Belgium

*Corresponding Author, Yuki Nishiguchi, M.A., Department of Cognitive and Behavioral Science, Graduate School of Arts and Sciences, The University of Tokyo, 3-8-1 Komaba, Meguro-ku, Tokyo 153-8902, Japan
Tel: +81-3-5454-6259
E-mail: ynishiguchi@beck.c.u-tokyo.ac.jp

Abstract

Previous studies have shown a negative correlation between effortful control (EC) and depressive symptoms. EC is defined as the efficiency of executive attention, which may be reduced by the attentional impairment associated with depression. However, the mechanism underlying this correlation is still unclear. We investigated the relationship between EC and depressive symptoms with the hypothesis that cognitive motivation, or need for cognition (NfC), is a possible mediator of this relationship. Participants were 178 Japanese university students. Each completed the Zung Self-Rating Depression Scale, Effortful Control Scale, and Need for Cognition Scale at baseline and follow-up assessments. Supporting our hypothesis, mediation analyses revealed a significant indirect effect of depressive symptoms on EC that was mediated by NfC. In addition, our data demonstrated a direct effect of depressive symptoms on EC. Longitudinal analysis indicated that an increase in depression and a decrease in NfC occurred synchronously, while NfC predicted an increase in EC over time. Depressive symptoms may decrease executive functioning and effortful control both directly and indirectly, the latter effect being mediated by motivation. These findings imply that a motivational deficit may partially explain the decreased EC found in people suffering from depression.

Keywords: depressive symptom, effortful control, need for cognition, executive function, negative mood, attention

The Need for Cognition Mediates and Moderates the Association between Depressive Symptoms and Impaired Effortful Control

1. Introduction

Recently, effortful control (EC; Rothbart et al., 2000) has received significant attention as a cognitive ability that could potentially prevent emotional disorders. EC is defined as the efficiency of executive attention, including the ability to plan, monitor errors, and inhibit a dominant response in favor of a subdominant response (Valiente et al., 2003). EC also enables individuals to monitor and resolve emotional conflicts. EC has been related to an ability to voluntarily focus and shift attention (Rothbart et al., 2000). Individuals with high EC show higher performance on attentional tasks (Rothbart et al., 2003).

A recent study reported a significant negative correlation between EC and depressive symptoms (Moriya and Tanno, 2008).This negative correlation may represent the attentional dysfunction, as impaired attentional control and attentional bias are associated with depression. The relationship between depression and attentional dysfunction has been investigated since Beck's (1967) cognitive theories of depression. Recent research has supported the idea that depression causes attentional bias, especially as regards disengagement of attention from negative information (see Koster et al., 2011). Such attentional bias can lead to the impaired regulation of negative emotion (MacLeod et al., 2002; Johnson, 2009; Sanchez et al., 2013). Additionally, as evidence in support of impaired control of attention in depression, individuals with higher level of depressive symptoms also exhibit decreased conflict resolution, including disturbances in attentional set-shifting (Austin et al., 2001; Rock et al., 2014) and lower performance on Stroop tasks (Paelecke-Habermann et al., 2005; Epp et al., 2012). Considering that these attentional abilities are strongly related to EC (Rothbart et al., 2000; Valiente et al., 2003), the negative correlation between EC and depressive symptoms possibly reflects impaired attentional control or attentional bias in

individuals with higher level of depressive symptoms.

Although some studies have reported a negative correlation between depressive symptoms and EC, the underlying mechanism that causes this negative correlation has not yet been investigated. To further characterize the relationship between EC and depressive symptoms, we introduced "need for cognition" (NfC; Cacioppo and Petty, 1982) as a potential mediating factor. NfC is a personality trait related to one's motivation to enjoy effortful cognitive activity (Cacioppo and Petty, 1982). Higher NfC leads individuals to direct more cognitive effort toward challenging cognitive processes like, for example, the internal conflict resolution required for some decision making (Bouckenooghe, et al., 2007). Moreover, some recent studies report that NfC affects cognitive functions requiring effort, including attention (Enge, et al., 2008; Fleischhauer et al., 2014). Since EC includes effortful attentional processes, we hypothesized a positive correlation between NfC and EC. Furthermore, based on these previous studies, we predicted that cognitive motivation (including NfC) may mediate the relationship between depressive symptoms and EC. For decades, studies have found a strong relationship between depressive symptoms and reduced motivation (e.g., Layne et al., 1982; Watson et al., 1995; Yang et al., 2014). Moreover, recent studies have indicated that the motivational deficits associated with depression can also lead to cognitive impairment, including deficits in attention (Austin, et al., 2001; Scheurich et al., 2008). Because increased motivation is thought to improve executive function (Pochon et al., 2002; Taylor et al., 2004; Banerjee et al., 2015), decreased motivation likely leads to impairments in cognitive ability. On the basis of these findings, we hypothesized that cognitive motivation mediates the relationship between depressive symptoms and EC.

Therefore, we investigated the possible mediational effect of NfC when introduced as a motivational factor. Depression has been shown to reduce motivation (Layne et al., 1982; Watson et al., 1995; Yang et al., 2014) and correlate negatively with NfC (Fleischhauer et al.,

2010), and so we predicted that NfC would correlate negatively with depressive symptoms and positively with EC. To our knowledge, the correlation between NfC and EC has not yet been studied. We expect that higher NfC increases EC because EC by definition includes effortful cognitive functions such as attentional control and internal conflict resolution.

We tested the moderation effect of NfC on the relationship between depressive symptoms and EC. If NfC strengthened EC, individuals with higher levels of NfC would be protected from EC impairment due to depressive symptoms. Thus, it was hypothesized that an interaction between depressive symptoms and NfC would be found, and a negative correlation between depressive symptoms and that EC would be stronger in individuals with lower NfC.

In addition to these cross-sectional analyses, we examined the longitudinal relationships among depressive symptoms, NfC, and EC. In the present study, we conducted a follow-up assessment on the same sample with an interval of approximately one month. First, we tested whether baseline levels of NfC were associated with subsequent increases or decreases in EC by computing residualized change scores of EC over the two assessments. Second, the prospective effects of depressive symptoms and NfC on EC, and vice versa, were tested using a cross-lagged model in the framework of path analysis (Finkel, 1985). We hypothesized that NfC would predict EC over time, and that EC might have prospective effects on NfC because individuals with higher EC are assumed to have executive attention that is more efficient, which can strengthen their motivation to engage in difficult cognitive activity. Moreover, it was expected that EC would have a negative effect on depressive symptoms, since EC is related to the regulation of negative emotionality (Moriya and Tanno, 2008).

2. Methods

2.1. Participants and Procedure

In the present study, undergraduate student volunteers at the University of Tokyo were recruited from a course on educational theory for the questionnaire-based survey. Before participating, all of them were told that their answers to the questionnaires would not influence their course grade and that all data would be processed anonymously. They did not receive any reward for their participation and they could freely decide to participate or not. After all information about the survey was given, participants signed to informed consent form if they wished to participate. The questionnaire-based survey was conducted two times during the course. A total of 178 students completed the initial assessment (T1) in the single session of the course. The mean age of the T1 sample was 19.2 (*SD* = 0.8). After 4 weeks, 115 students from T1 sample completed the second assessment (T2). The mean age of the T2 sample was 19.2 (*SD* = 0.8), and all of them were tested in the same class. In T1 and T2, the same questionnaire measures were used, and participants finished the questionnaire in about 10 minutes both times.

2.2. Measures

2.2.1 Zung self-rating depression scale (SDS; Zung, 1965). The Japanese version of the SDS (Fukuda and Kobayashi, 1973) was used to measure depressive symptoms. This scale is composed of 20 items (e.g., "I feel down-hearted and blue.") asking how they had felt during the preceding week. All items are rated on a 4-point scale, ranging from (1) "a little of the time" to (4) "most of the time." The average SDS score was 43.11 at T1 and 43.51 at T2 (Table 1). These scores were similar to previously reported averages on the SDS (41.58, Sakamoto et al., 1998) for a sample of Japanese undergraduate students. The SDS cutoff score of 50 often has been used to identify clinically relevant levels of depressive symptoms; and in the present study, 20.79% of the T1 sample and 18.26% of the T2 sample scored above 50.

2.2.2 Adult version of the effortful control scale (Rothbart et al, 2000). The

Japanese version of the effortful control scale (Yamagata et al., 2005) was used to measure EC. This scale is composed of 35 items rated on a 4-point scale ranging from (1) "not true of you" to (4) "true of you." This scale is made up of three subscales: inhibitory control (11 items; e.g., "Even when I feel energized, I can usually sit still without much trouble if it's necessary."), activation control (12 items; e.g., "As soon as I have decided upon a difficult plan of action, I begin to carry it out."), and attention control (12 items; e.g., "I am usually pretty good at keeping track of several things that are happening around me."). Inhibitory control represents the ability to suppress inadequate behavior. Activation control is a function which enables one to perform an action despite a strong tendency to avoid it. Attentional control is the capacity to focus and shift attention when and where desired. The total of these subscale scores is the EC score, which indicates overall efficiency of EC.

2.2.3 Need for cognition scale (Cacioppo and Petty, 1982). The Japanese version of the need for cognition scale developed by Kouyama and Fujihara (1991) was used to quantify NfC. This scale has 15 items (e.g., "I find it especially satisfying to complete an important task that required a lot of thinking and mental effort.") rated on a 7-point scale, ranging from (1) "strongly disagree" to (4) "strongly agree."

2.4. Data analyzing procedure

Data analyses were performed by using the Statistical Package for Social Sciences (SPSS, version 22.0) computer software and R (R 3.2.0, R Development Core Team) with sem package (Fox et al., 2015). As cross-sectional analyses, we computed descriptive statistics and correlations between depressive symptoms, EC, and NfC. Second, multiple regression analyses were conducted according to Barron and Kenny's (1986) framework in order to identify any mediation effects. Subsequently, indirect effects were analyzed with the bias corrected bootstrapping methods (10,000 bootstrap samples, 95% confidence intervals) suggested by Preacher and Hayes (2008) because bootstrapping method is thought to prevent

problems caused by non-normal distribution of indirect effects (Preacher et al., 2007), thus it is more powerful than Sobel test (Sobel, 1982) that is commonly used for testing indirect effect. Third, the moderation effect of NfC on the relationship between depressive symptoms and EC was analyzed using multiple regression. In the regression model, the EC scores were predicted by depressive symptoms, NfC, and the interaction between depressive symptoms and NfC. In the moderated regression analysis, depressive symptom and NfC scores were mean-centered before computing the interaction term. The interaction of depressive symptoms and NfC was analyzed further using simple slope analysis (Aiken and West, 1991).

Longitudinal data were analyzed using two approaches. First, we computed the residualized change scores of each variable, which were assumed to be independent of the T1 scores of the measures (Cohen et al., 2003). The residualized change scores were calculated using simple linear regression models, wherein T2 scores were predicted by T1 scores. By using these change scores, we examined the correlations between baseline and the change at the follow-up assessment of depressive symptoms, NfC, and EC. Second, structural equation modeling was used to analyze the effects of the T1 scores on the T2 scores. In the present model, it was hypothesized that NfC at T1 would predict EC at T2, and that EC would predict NfC at T2 and depressive symptoms at T2.

3. Results

3.1 General findings

Table 1 presents the descriptive statistics of all self-report measures assessed in T1 and T2. All correlations between depressive symptoms, EC, EC subscales and NfC were found to be significant (Table 1; $p < 0.05$).

3.2 Mediation effects

First, cross-sectional mediation analysis was conducted on T1 data. Following Baron and Kenny's steps with total EC score as the dependent variable, multiple regression analyses

were conducted (Figure 1). The first equation regressed EC on depressive symptoms (β = -0.55, t = -8.65, $p < 0.001$), the second regressed NfC on depressive symptoms (β = -0.29, t = -4.06, $p < 0.001$), and the third regressed EC on both depressive symptoms and NfC ($F(2,175) = 46.03$, $R^2 = 0.34$, $p < 0.001$). Controlling for the effect of depressive symptoms, the effect of NfC on EC was significant (β = 0.23, t = 3.51, $p < 0.001$). Furthermore, the effect of depressive symptoms on EC remained even when the effect of NfC on EC was controlled for (β = -0.48, t = 7.51, $p < 0.001$). These results met the requirements for a partial mediation effect (Baron and Kenny, 1986).

Next, the indirect effect of depressive symptoms on EC through the mediation of NfC was estimated with bias corrected bootstrapping according to Preacher and Hayes (2008). Bootstrapping analyses revealed a significant indirect effect of depressive symptoms on EC through the mediation of NfC (point estimate = -0.11; 95% CI [-0.23, -0.03] was entirely below zero). We conducted similar follow-up analyses (10,000 bootstrap samples and 95% CI) with each EC subscale (inhibitory control, activation control & attention control) as the dependent variable. Results revealed a significant negative indirect effect of depressive symptoms on all subscales: inhibition control (point estimate = -0.03; 95% CI = [-0.07, -0.01]), activation control (point estimate = -0.04; 95% CI = [-0.09, -0.00]) and attention control (point estimate = -0.04; 95% CI = [-0.09, -0.01]). These results indicate that depressive symptoms had similar indirect effect on all aspects of EC.

On the same sample, we also tested the reverse model (EC affects depressive symptoms through the indirect effect mediated by NfC) to investigate the possible effect of EC on depressive symptoms. The first equation regressed depressive symptoms on EC (β = -0.55, t = -8.65, $p < 0.001$), the second regressed NfC on EC (β = 0.37, t = 5.21, $p < .0001$), and the third regressed depressive symptoms on both EC and NfC ($F(2,175) = 39.02$, R^2 = 0.30, $p < 0.001$). Controlling for the effect of EC, the effect of NfC on depressive symptoms

was not significant (β = 0.11, t = -1.59, p = *n.s.*). The effect of EC on depressive symptoms remained significant when the effect of NfC on depressive symptoms was controlled for (β = -0.51, t = 7.51, $p < 0.001$). These results do not meet the requirements for a mediation effect (Baron and Kenny, 1986). The indirect effect was also estimated for the reverse model with bootstrapping analysis, however the indirect effect of EC on depressive symptoms through the mediation of NfC was not significant (point estimate = -0.02; 95% CI [-0.06, 0.00] includes zero).

3.3 Moderating effects

We tested the moderating effect of NfC on the negative association between depressive symptoms and EC using multiple regression. Because NfC had been hypothesized to reinforce the efficiency of attentional functioning, individuals with higher levels of NfC were expected to be less likely to suffer from cognitive impairment caused by depressive symptoms. In the regression model, T1 depressive symptoms, NfC, and their interaction predicted T1 EC. The regression model explained a significant amount of the variance of EC (F(3,174) = 34.07, R^2 = 0.36, $p < 0.001$). Each of the predictors, depressive symptoms (B = -0.79, SE = 0.10, t = -7.61, $p < 0.001$), NfC (B = 0.17, SE = 0.06, t = 2.96, $p < 0.01$), and their interaction (B = 0.02, SE = 0.01, t = 2.65, $p < 0.01$), significantly predicted EC. In order to explore the form of this interaction, we conducted a simple slope analysis by calculating the conditional effects of depressive symptoms on EC for higher and lower levels of NfC (mean ± 1*SD*; Aiken & West, 1991). As shown in Figure 2, the conditional effect was stronger for lower (B = -1.04, SE = 0.14, t = -7.42, $p < 0.001$) than for higher levels of NfC (B = -0.53, SE = 0.14, t = -3.77, $p < 0.001$). These results imply that NfC has a protective effect that ameliorates the negative effect of depressive symptoms on attentional functions.

3.4 Longitudinal analyses

We tested the present data for longitudinal effects. First, we tested whether the T1

scores of the self-report measures correlated with the change scores of the measures. We used the residualized change scores as an index of change. Correlation analysis showed that the T1 depressive symptoms were not significantly correlated with the residualized change scores of NfC (r = -0.13, p = *n.s.*) or EC (r = -0.03, p = *n.s.*). The NfC scores at T1 were significantly correlated with the residualized change scores of EC (r = 0.23, $p < 0.05$) but not with the residualized change scores of depressive symptoms (r = -0.15, p = *n.s.*). The EC scores at T1were significantly and negatively correlated with the residualized change scores of depressive symptoms (r = -0.18, $p < 0.05$), and with residualized change scores of NfC (r = 0.22, $p < 0.05$). Overall, these findings indicate that T1 NfC was positively associated with increased EC and that EC at T1 was positively associated with increased NfC; thus, NfC and EC may positively interact over time. On the other hand, T1 EC was negatively associated with changes in depressive symptoms, which may indicate that higher EC protects individuals from increases in depressive symptoms.

Next, the longitudinal effect was tested using path analysis. Following the results of the change score analysis, a model in which NfC at T1 predicted EC at T2, and EC at T1 predicted NfC T2 and T2 depressive symptoms (Figure 3), showed an excellent fit to the data ($\chi^2(3) = 1.83$, p = *n.s.*; GFI = 0.99, AGFI = 0.96, RMSEA = 0.00). The T1 depressive symptoms were positively and significantly associated with T2 depressive symptoms (β = 0.70, $p < 0.01$). NfC at T1showed a significant positive association with NfC at T2 (β = 0.73, $p < 0.01$) and with EC at T2 (β = 0.11, $p < 0.05$), which suggests that individuals with higher NfC at T1, had higher EC at T2. EC at T1 showed a significant positive association with EC at T2 (β = 0.82, $p < 0.01$) and T2 NfC (β = 0.19, $p < 0.01$); and had a significant negative association with T2 depressive symptoms (β = -0.16, $p < 0.05$). The results of the structural equation modeling were similar to those of the change score analyses, which suggest a positive interaction between NfC and EC, and a buffering effect of EC on depressive

symptoms.

4. Discussion

The present study investigated the mediating effects of NfC on the relationship between depressive symptoms and EC. There are three novel findings. First, our results showed a positive correlation between NfC and EC, which has not been shown by previous studies. Second, we revealed that the effect of depressive symptoms on EC was partially mediated by cognitive motivation (NfC). The present results support our hypothesized mediational model in which NfC mediates the effect of depressive symptoms on EC. The results of our bootstrap analyses indicate a significant indirect effect of depressive symptoms on EC, mediated by NfC. Our results indicated only a partial mediation: the direct effect of depressive symptoms on EC was still significant after controlling for the effect of NfC. Thus, our results suggest two different effects of depressive symptoms on EC: one is a direct effect and the other is an indirect effect that is mediated by NfC. Third, the present study revealed the moderating effect of NfC on the association between depressive symptoms and EC, which indicated that higher NfC protects EC against negative effect caused by depressive symptoms.

A comparison of our results with those of previous studies reveals some important implications about the mechanisms underlying these two effects. We begin by discussing the simple effects of depressive symptoms on NfC and NfC on EC in turn. First, we introduced NfC in our study as a motivational factor and found a negative correlation between it and depressive symptoms, consistent with previous findings (Fleischhauer et al., 2010). Considering that NfC is a personality trait strongly related to cognitive motivation, this association is consistent with the literature on reduced motivation in depression (Layne et al., 1982; Watson et al., 1995; Yang et al., 2014). Many previous studies support the notion that depression reduces motivation (Layne et al., 1982; Watson et al., 1995; Yang et al., 2014). In

particular, NfC is strongly related to an interest in complex cognitive activity (Cacioppo and Petty, 1982), and the loss of interest correlated with depressed mood may be associated the decline in NfC.

Second, our results revealed a significant positive correlation between NfC and EC. Previous studies have shown that NfC may influence attention (Enge et al., 2008; Fleischhauer et al., 2014), and our results extend this to show that NfC is associated with increased EC efficiency. Previous study indicated that motivation affects the dorsolateral prefrontal cortex, which is strongly related to the control of cognitive function, including attention, through the anterior cingulate cortex (Pessoa, 2009). Since motivation may improve attention, it may also increase EC, which includes attentional abilities. Interestingly, our results showed that the indirect effect of depressive symptoms was significant for all EC subscales. Since inhibition control, activation control and attentional control all require some cognitive effort, the decline of NfC associated with depressive symptoms may predict less efficiency of all aspects of EC. Altogether, the indirect effect observed in this study appears to have been produced by the combination of the positive effect of NfC on EC and the negative effect of depressive symptoms on NfC. This indirect effect, however, may not entirely be explained by the mediation of motivation. For example, the negative attentional bias associated with depression or social anxiety, which is thought to occur involuntarily, may disrupt EC (Moriya and Tanno, 2008). Considering the partial effect of NfC on EC, involuntary attentional processes that are largely unaffected by motivation may also have a substantial effect on EC.

The direct effect of depressive symptoms on EC can be explained by the effect of emotional state on affective perception. According to Pessoa (2009), emotional state can enhance the affective significance of information. For example, an anxious state makes the emotional impact of threat stimuli larger, which is emotionally relevant to anxiety. This

system influences the perception of affective stimuli and can modify executive control functions like selective attention. These emotional changes to executive function may manifest as an impairment of attentional control, as is the case with depression's biased processing and difficulty on disengaging attention from negative stimuli (Koster, et al., 2005; Sears et al., 2010). Since EC is related to emotional control (Kochanska et al., 2000; Kanske and Kotz, 2012), such a dysfunction of emotional regulation may have resulted in the lower EC scores observed in the present study, though this cannot be concluded, because the present study did not include the assessment of attentional bias.

Supplementary, the reversed model, in which EC affects depressive symptoms through the indirect effect mediated by NfC, was also tested in the present study. Although the direct effect of EC on depressive symptoms was found to be significant, the indirect effect of EC on depressive symptoms through the mediation of NfC was not significant. Thus, the reversed indirect effect of EC on depressive symptoms was not supported in the present study, though the reversed direct effect was observed.

Additionally, the present results revealed several important findings. Moderation analysis showed that NfC had a moderating effect, which protects individuals from lower EC related to depressive symptoms. The moderation analysis showed that individuals with lower NfC tended to suffer from lower EC when they had depressive symptoms. NfC was related to intrinsic motivation (Cacioppo and Petty, 1986), and intrinsic motivation was associated with well-being (Kasser and Ryan, 1993; Ryan and Deci, 2000). Our results may explain the association between intrinsic motivation and well-being, in that the individuals with higher intrinsic cognitive motivation maintained more efficient EC, even when they had depressive symptoms, which may have helped them regulate negative emotionality.

Our short-term longitudinal data indicated that baseline NfC predicted increased EC at the follow-up assessment of both the in residualized change and the cross-lagged model

analyses. We also found a significant reversed effect: higher EC at T1predicted higher NfC at T2. These results suggest a bidirectional relationship between NfC and EC, in which motivation enhances attentional functioning, and vice versa. Furthermore, the results of the path analysis indicated that higher EC at T1 was associated with a reduction of depressive symptoms at T2. This ameliorating effect of EC on subsequent depressive symptoms is congruent with previous findings that EC is associated with the efficient regulation of negative emotionality (Moriya and Tanno 2008).

Three points should be clarified in future research. First, in this study, we only considered the effect of depressive symptoms on EC and did not test the effects of other emotional disturbances or types of motivational factors other than NfC. There is considerable evidence that other emotional disorders (e.g., social anxiety) also correlate with EC (e.g., Moriya and Tanno, 2008). Future studies should also control for other factors such as anxiety or social anxiety because anxiety, social anxiety, and depression frequently correlate with one another. Moriya and Tanno (2008) also showed that controlling for social anxiety resulted in a no-longer significant correlation between depressive symptoms and the attentional control subscale of EC, while the correlations between depressive symptoms and inhibition control, activation control, and overall EC score were all still significant. Therefore, controlling for other emotional disturbances may lead to different patterns of results for each EC subscale. In addition, only NfC was assessed as a motivational factor in the present study because NfC was related to attention in previous studies, however the effect of other type of motivation was not considered. To specify the distinctive effect of depressive symptoms on EC, more related factors should be assessed with larger sample in future studies.

Second, the present study included short-term longitudinal data, however, sample size was limited and interval between two surveys was not long enough to argue causal

relationships between traits. For a closer investigation on longitudinal interaction, assessments on larger samples and over multiple years are required.

Third, the present study was conducted with a non-clinical sample, and it is unclear whether the present findings can be applied to individuals diagnosed with depression. Since previous results have indicated that there is a motivational deficit in individuals with major depression (Layne et al., 1982), it is possible that individuals diagnosed with depression also show impaired EC caused by NfC, in the same way as the present model showed. As discussed earlier, the present model also found a moderating effect of NfC; thus, individuals with clinical depression suffer from impaired EC, especially when they have lower NfC. However, replication with a clinical sample must be conducted to draw a definitive conclusion.

4.1 Conclusions

The relationship between depressive symptoms and EC is partially mediated by NfC. Our results indicate that EC is affected by depressive symptoms in two ways. First, EC is impaired by the decreased motivation brought on by depressed mood. Second, elevated depressive symptoms directly predict decreased EC. These two pathways could be supporting evidence of the theories proposing that attention is influenced by motivation and emotion.

Although most recent studies on attention in depression have focused on the negative attentional bias, our results indicate the importance of motivation to attention. Future studies of attention in depressed subjects should explore whether individuals with higher level of depressive symptoms experience dysfunction in effortful processes of attention, such as the top-down control of attention or conflict resolution. Additionally, our results have implications for future research on interventions targeted at the cognitive deficits of depression. Since motivational impairment leads to the attentional impairment in individuals

with depressive symptoms, procedures that require more cognitive effort may be less effective as a method of improving attentional control. In other words, procedures which do not require much cognitive effort to improve cognitive deficits (e.g., cognitive bias modification; see Hertel & Mathews, 2011) may be especially effective. The present data also showed a positive effect of EC on NfC over time, improving executive attention by attentional training (Wells, 1990) or mindfulness training (Mindfulness based stress reduction, Kabat-Zinn, 1990) possibly affect NfC positively.

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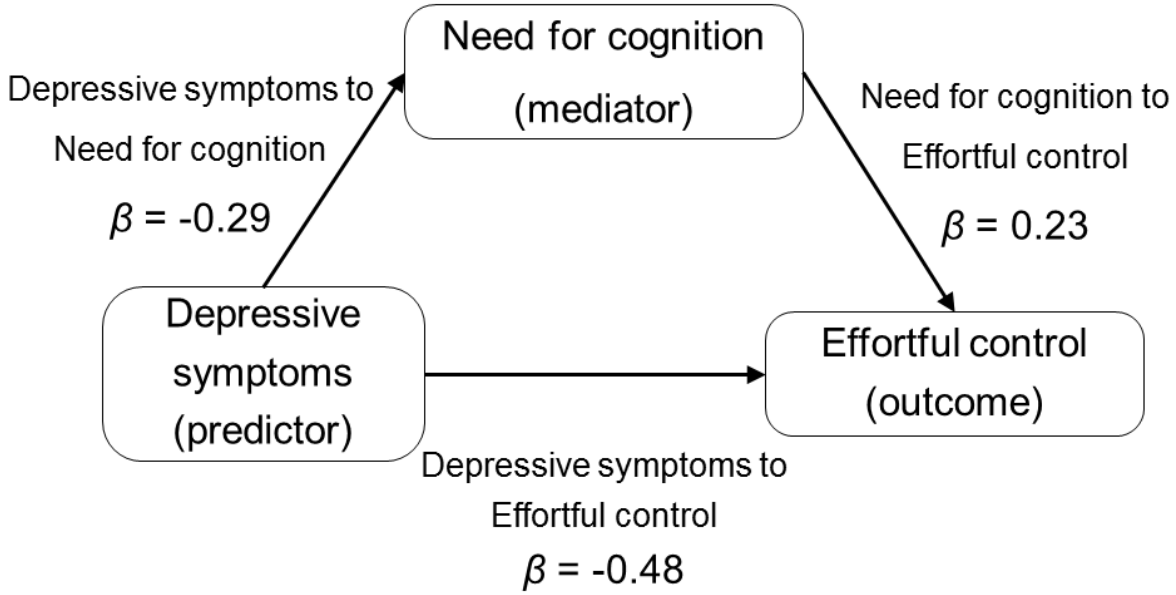
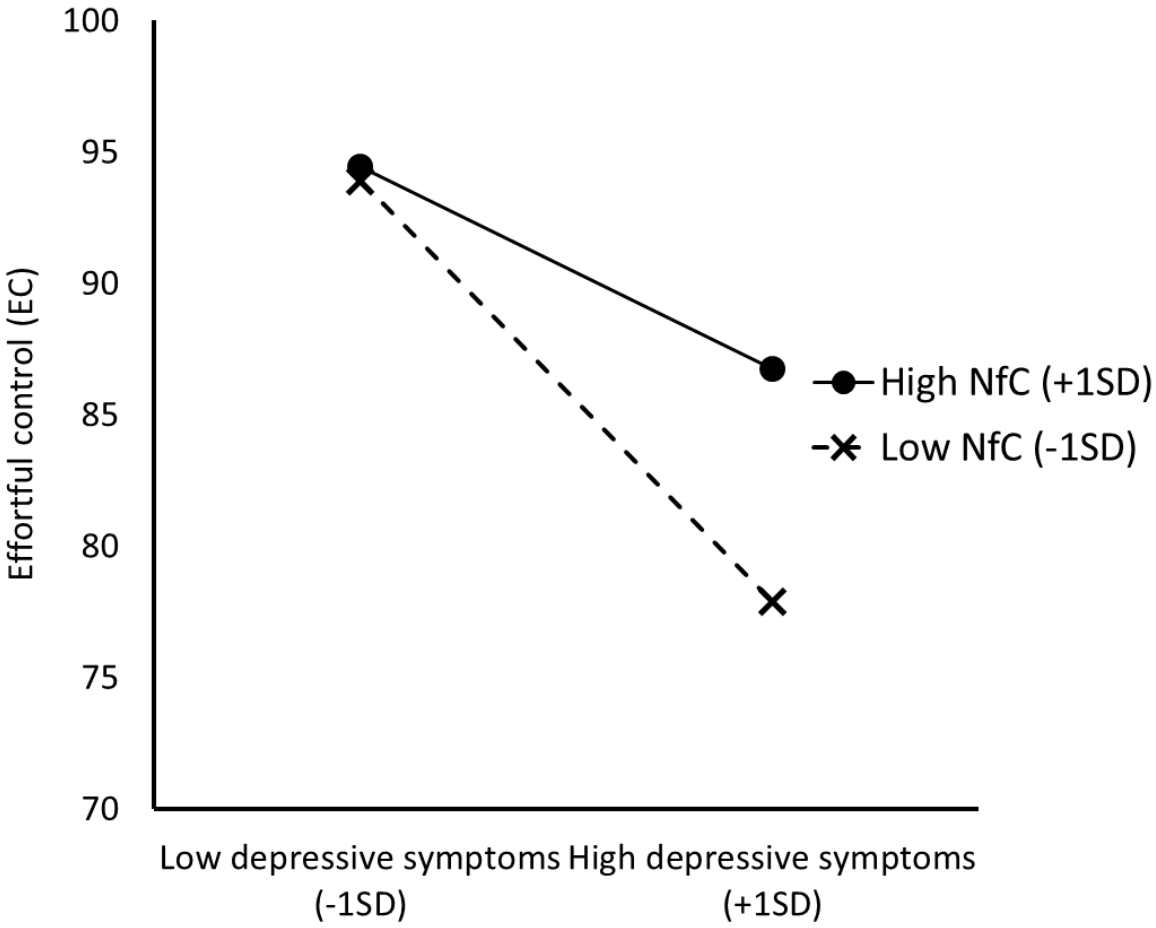
Figure captions

Figure 1. All coefficients shown in this figure are estimated by T1 data. NfC mediates the effect of depression on EC. Regression analyses showed that all pathways were significant ($p < 0.01$). In the same way, we conducted mediation analysis with T2 data for replication. The results of T1 data was successfully replicated, and all the coefficients were also significant ($p < 0.01$) with T2 data. Indirect effect was also significant with T2 data (95%CI = [-0.30, -0.02]).

Figure 2. Scores of Effortful control scale was predicted by depressive symptoms at high and low level of NfC. EC was more strongly affected by depressive symptoms when NfC was lower.

Figure 3. Estimated model of depressive symptoms, EC and NfC at T1 and T2. *Note*; ** = $p < 0.01$, * = $p < 0.05$, † = $p < 0.10$, All coefficients are standardized.





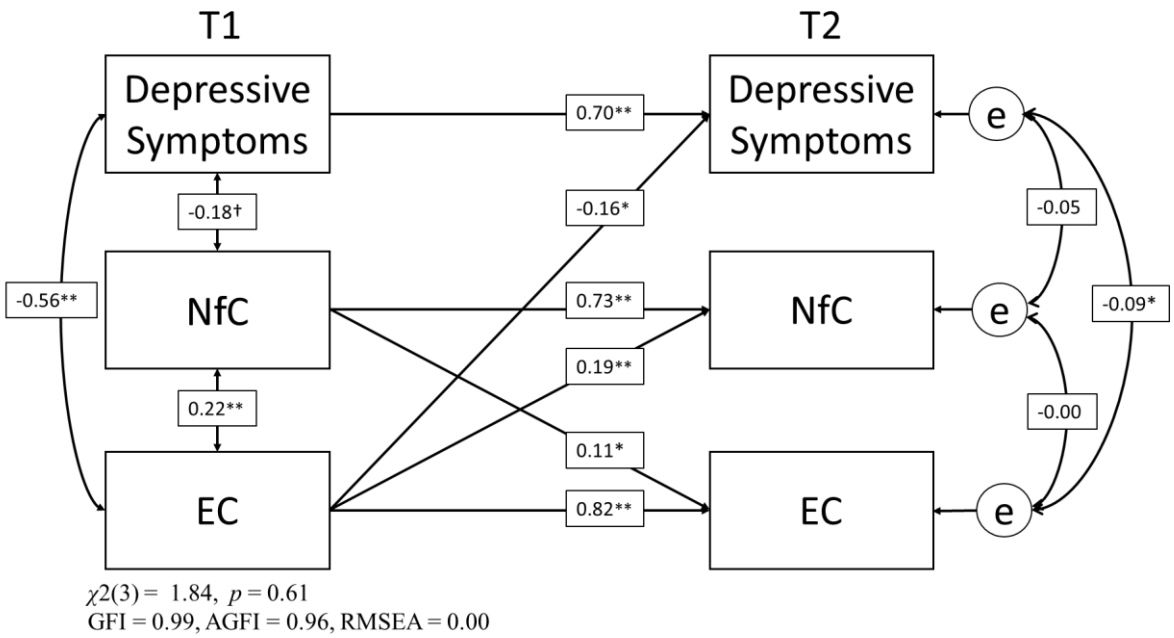


Table 1. Pearson correlation coefficients between self-report measures

	1	2	3	4	5	6	7	8	9	10	11	*Mean*	*SD*	*α*
1. Depressive symptoms -T1	1.00											43.11	7.50	0.82
2. Effortful control -T1	-0.55**	1.00										87.70	12.33	0.86
3. Inhibitory control -T1	-0.31**	0.71**	1.00									29.87	4.15	0.58
4. Activation control -T1	-0.50**	0.83**	0.43**	1.00								29.16	6.14	0.83
5. Attention control -T1	-0.44**	0.78**	0.36**	0.42**	1.00							28.67	5.52	0.79
6. Need for cognition -T1	-0.29**	0.37**	0.27**	0.29**	0.29**	1.00						64.85	13.83	0.84
7. Depressive symptoms -T2	0.80**	-0.56**	-0.37**	-0.51**	-0.46**	-0.24*	1.00					43.51	7.23	0.83
8. Effortful control -T2	-0.49**	0.85**	0.64**	0.73**	0.68**	0.31**	-0.58**	1.00				86.93	12.71	0.89
9. Inhibitory control -T2	-0.23*	0.58**	0.72**	0.41**	0.35**	0.22*	-0.30**	0.71**	1.00			29.69	3.96	0.66
10. Activation control -T2	-0.45**	0.80**	0.54**	0.86**	0.51**	0.22*	-0.53**	0.87**	0.47**	1.00		29.14	6.33	0.85
11. Attention	-0.45**	0.62**	0.34**	0.40**	0.74**	0.31**	-0.52**	0.81**	0.38**	0.52**	1.00	28.10	5.45	0.83

control -T2														
12. Need for cognition -T2	-0.23*	0.35**	0.20*	0.29**	0.35**	0.78**	-0.31**	0.39**	0.28**	0.33**	0.32**	64.86	15.13	0.89

Note. Correlations between T1 scores were calculated on all undergraduates who participated in the T1 survey (N = 178). Correlations between T1 and T2 scores were calculated on participants who completed both surveys (N = 115). * = $p < 0.05$; ** = $p < 0.01$

- **Highlights**
- ·Depressive symptoms predicted a decrease in EC through the mediation of NfC.
- ·Elevated depressive symptoms and decreased NfC occurred synchronously.
- ·NfC significantly predicted an increase in EC over time.
- ·Depressive symptoms predicted lower EC via motivational and non-motivational paths.